



## Septic shock as the primary presentation of missed traumatic intraperitoneal bladder rupture: A brief clinical report

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### Abstract

Intraperitoneal bladder rupture after blunt abdominal trauma is uncommon and may be missed on initial imaging. We report a middle-aged man who presented in septic shock two days after a seemingly trivial fall. Contrast enhanced CT abdomen demonstrated only intraperitoneal free fluid without solid organ injury. Despite resuscitation, he developed worsening peritonitis, oliguria, and escalating vasopressor requirements. Exploratory laparotomy performed on clinical grounds revealed a full thickness posterior bladder perforation with approximately 1L of urine in the peritoneal cavity. The bladder was repaired, and the patient required postoperative ventilation, vasopressor support, and hemodialysis for sepsis induced acute kidney injury. He recovered fully and was discharged on postoperative day 25. This case highlights that traumatic bladder rupture may present primarily with septic shock and nondiagnostic imaging, and that timely surgical exploration based on clinical judgment is lifesaving.

### Introduction

Urinary bladder rupture following Blunt Abdominal Trauma (BAT) is rare, with an incidence of 0.3–1.6% [1]. Intraperitoneal (IP) ruptures are less common than extraperitoneal injuries but carry greater morbidity and typically require operative repair [2]. Although Contrast Enhanced CT (CECT) with delayed images or CT cystography is highly sensitive [3], missed injuries continue to be reported [4]. Diagnostic uncertainty increases when free intraperitoneal fluid is present without solid organ injury, particularly in patients with chronic alcoholism where ascites may be presumed. Septic shock as the primary presentation of traumatic bladder rupture is exceptionally uncommon [5]. We present a case in which clinical deterioration despite inconclusive imaging prompted timely laparotomy and diagnosis.

### Case report

A 45 yr old man with chronic alcoholism fell while walking two days before presentation. He developed progressive abdominal pain and oliguria following the fall and was admitted to a local hospital, where norepinephrine infusion was initiated for worsening shock. With escalating vasopressor requirements, he was transferred to our tertiary center.

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On arrival, he was tachycardic (135/min), tachypneic (36/min), and hypotensive (100/64 mmHg) despite norepinephrine at 8 mL/h. The abdomen was distended with generalized rebound tenderness. Urine output was minimal, and blood-tinged urine was noted in the Foley catheter. Outside laboratory reports suggested acute kidney injury and possible septic shock.

CECT abdomen revealed moderate intraperitoneal free fluid without solid organ injury, pneumoperitoneum, or other pathology. The fluid did not show blood attenuation and was initially interpreted as ascites. Arterial blood gas analysis showed severe metabolic acidosis (pH 7.1) and mildly elevated lactate (3.9 mmol/L).

In the ICU, vasopressor requirements continued to rise, and so vasopressin was added. Liver function tests were grossly normal, and INR was mildly elevated (1.7). Given the patient's alcoholism and the presence of free fluid, the initial working diagnosis was decompensated liver disease with sepsis. However, persistent peritonitis, oliguria, unexplained haematuria, and worsening shock despite resuscitation raised concern for missed intra-abdominal injury.



After multidisciplinary discussion, exploratory laparotomy was performed. Approximately 1 L of turbid, straw-coloured fluid with a urine like odour was found. A full thickness perforation was identified on the posterior bladder wall, consistent with intraperitoneal rupture. No other injuries were present. The bladder was repaired in two layers, and peritoneal lavage was performed. Fluid creatinine confirmed urinary ascites.

Postoperatively, the patient required mechanical ventilation for seven days and vasopressor support for 48 hours. Hemodialysis was initiated for sepsis induced acute kidney injury (KDIGO stage 3), requiring ten sessions. Paralytic ileus resolved by Postoperative Day (POD) 10. Wound dehiscence on POD 12 required secondary suturing. Renal function gradually improved, and the Foley catheter was removed on POD 15. He was discharged on POD 25 in stable condition.

### Discussion

Bladder injuries occur in approximately 1.6% of BAT cases [2]. Intraperitoneal ruptures typically require operative repair due to the risk of urinary peritonitis and chemical inflammation. Although urine is sterile, prolonged intraperitoneal contamination can lead to sepsis and, rarely, septic shock [5].

CECT is the investigation of choice for BAT, yet missed injuries remain a challenge. In this case, free fluid without solid organ injury was initially interpreted as ascites, delaying recognition of bladder rupture. Similar diagnostic dilemmas have been described, particularly in patients with chronic alcoholism or minimal trauma history [4].

Earlier EAST guidelines recommended exploratory laparotomy in BAT patients with free fluid and no solid organ injury due to concern for hollow viscus injury [6]. More recent recommendations from the Western Trauma Association and WSES emphasize individualized decision making, including observation, serial examinations, diagnostic laparoscopy, or laparotomy depending on physiologic status [7].

In our patient, persistent peritonitis, escalating vasopressor requirements, oliguria, and hematuria mandated surgical exploration despite nondiagnostic imaging. This aligns with current recommendations emphasizing clinical judgment over imaging when physiologic instability persists.

Fluid analysis for creatinine can aid diagnosis when feasible; a peritoneal fluid creatinine level three times higher than serum suggests urinary leakage [5]. In this case, the patient's critical condition precluded preoperative sampling.

Septic shock as the primary presentation of traumatic intraperitoneal bladder rupture is rare. Most reports involve spontaneous or postoperative ruptures [5]. This case adds to the limited literature describing traumatic bladder rupture manifesting predominantly with septic shock and nondiagnostic CT findings.

### Conclusion

Traumatic intraperitoneal bladder rupture may present atypically and can be missed on initial imaging. In BAT patients with intraperitoneal free fluid, oliguria, hematuria, and persistent hemodynamic instability, bladder injury should be strongly considered even when CT is inconclusive. Early surgical intervention based on clinical judgment is crucial and may be lifesaving.

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